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ABSTRACT

The relationship between low elevated lead absorption and mild mental retardation was investigated in 40 rural children (preschool to grade 12) without demonstrable cause for their retardation. Trace mineral analysis of hair samples from Ss and a control group (N=20) indicated the mean hair lead concentrations for the retarded Ss were considerably higher than those of control Ss. Potential sources of lead included deteriorating paint chips, illicitly distilled whiskey, and exposure through fertilizers and pesticides containing lead. (CL)

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LOW ELEVATED LEAD LEVELS AND MENTAL RETARDATION

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"Low Elevated Lead Burdens
and Mental Retardation"

This study examined a sample of Appalachian children with mild mental retardation for the presence of previously unidentified lead poisoning or elevated body lead levels. Children who survive lead poisoning are at risk for severe mental retardation resulting from damage to the central nervous system (Byers & Lord, 1943). More recent research has suggested that children with a moderate increase in body burden of lead, although clinically asymptomatic, may also be subject to impaired mental development, albeit a more difficult type to define. Low elevated lead levels previously thought harmless are now being associated with mild mental retardation and impaired IQ (David et al., 1976; de la Burde & Choate, 1975; Needleman et al., 1979; Routh et al., 1979).

Undue exposure to lead is a major pediatric health problem nationwide. Data from the Environmental Health Services Division, Center for Disease Control, indicate that from 1973 to 1978, among 2,380,942 children screened by projects funded under the Lead Based Paint Poisoning Prevention Act, 162,580 had elevated lead levels; 20,994 were diagnosed as lead poisoned. Since nationwide screening began, lead poisoning in children has declined, but elevated lead burdens remain prevalent. Some urban areas report the condition in 15 percent of the children screened (Lin-Fu, 1979). Although conceived as primarily an urban concern, children living in rural areas are not spared from this

health problem, although the prevalence appears to be lower. A survey of 230 rural children from Dutchess County, Conneticut revealed that nine percent had elevated lead burdens (Lepow, 1972).

The purpose of this study was to investigate the relation between low elevated lead absorption and mild mental retardation in children without demonstrable cause for their mental deficit. The subjects were drawn from the Upper Cumberland Region of Tennessee. We are not aware of any previous studies which have examined mildly retarded Appalachian children for the presence of increased lead burdens. The following hypothesis was proposed: the group of retarded children aetiology unknown would have lead burdens significantly higher than a control sample.

Methods

Subjects

The 67 subjects in this study were drawn from schools in the Tennessee counties of Jackson (N=16), Overton (N=4), Putnam (N=8), and Scott (N=39). These schools included a preschool developmental center, four elementary schools, and two high schools (grades 7-12).

All of the mentally retarded subjects were receiving special educational services. Their diagnosis of retardation was based on an overall evaluation from a series of consultations by school psychologists, classroom teachers, and appropriate other specialists where indicated. The diagnostic

evaluation included the administration of a standardized intelligence test. All of the retarded children in the sample had an IQ of 55-84 inclusive (The American Association of Mental Deficiency classifies an IQ of 55-69 as "mild mental retardation" and 70-84 as the "borderline range.")

Presence or Absence of Probable Cause

All of the children's school records which met the IQ criteria were reviewed to determine whether there was a known or highly probable medical reason for their retardation. On the basis of this review, children with "probable cause," e.g., Down's Syndrome, hydrocephaly, brain injury, were removed from the study. In addition, any child with a history of diagnosed lead poisoning was excluded from the sample study. Only children with unremarkable medical histories for mental retardation were assigned to the "aetiology unknown" group (N=40).

The control subjects (N=27) were randomly drawn from the general school population at the six schools. No IQ determinations were available for the normal comparison group, but it appears safe to assume a relative absence of mental retardation in a randomly drawn general school population. Furthermore, a survey of the teachers' reports obtained for each child indicated average academic achievement and learning ability.

Table 1 shows the relevant demographic data for the two resulting groups of subjects. There were no significant

differences between the groups in age, socio-economic status, or in the sex and ethnic group distributions.

Insert Table 1 about here

Classification of Lead Levels

After obtaining parental permission, children were asked to submit a small sample of hair (about 40 mg.) for trace mineral analysis. Hair samples were collected by the researchers using stainless steel scissors and then submitted to Doctor's Data, Inc., a federally licensed hair analysis laboratory in West Chicago, Illinois, where they were analyzed by atomic absorption spectroscopy.

Scalp hair has several characteristics of an ideal tissue for epidemiologic study in that it is painlessly removed, normally discarded, easily collected, and its contents can be analyzed relatively easily (Hammer, Finklea, Hendricks, Shy & Horton, 1971). Trace elements, in particular, are accumulated in hair at concentrations that are generally at least ten times higher than those present in blood serum, provide a continuous record of nutritional status and exposure to heavy metal pollutants and may serve as a probe of physiologic functions (Maugh, 1978).

The best results have been obtained with heavy metal pollutants such as lead. Numerous investigations world wide have shown that concentrations of lead in the hair provide an

accurate and relatively permanent record of exposure, and that there is a strong correlation between concentrations in hair and concentrations in internal organs (Kyle & Pease, 1966; Schroeder & Nason, 1969).

Results

The two groups of children were compared for hair-lead concentrations. As shown in Table 2 the mean concentrations for the retarded children with unknown aetiology were

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Insert Table 2 about here

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considerably above those of the control children. The retarded group had a mean hair-lead of 15.9 ppm. while the control group had a mean hair-lead of 9.16. The data was analyzed with the *t*-test for two independent samples design, yielding a statistically significant *t* value, (t= 3.39, 65, $p < .001$) indicating that the variation between the two means was unlikely to have occurred by chance.

Fifteen ppm. is considered to be the normally tolerated limit of hair lead (Doctor's Data, 1980). The distribution of lead concentrations in the two groups is shown in Table 3.

The retarded group had the largest number of children in the higher ranges, 15 ppm. and above. Forty-five percent of the retarded children had lead concentrations above the normally tolerated limit compared to approximately 9% in the

control group. No children in this study showed extreme concentrations associated with lead poisoning.

Insert Table 3 about here

Discussion

While not establishing an aetiological relationship, the data of this study shows a clear association between elevated hair lead concentrations and mild mental retardation in rural children. The study's hypothesis was strongly supported by the data.

The confidence with which mild mental retardation can be attributed to past lead exposure depends upon the adequate classification of earlier lead exposure. The classification of lead exposure to hair lead levels has been validated in a number of earlier studies. Hair lead levels are elevated in children with chronic lead poisoning (Kopito, Byers, & Shwachman, 1967) and in children with increased lead burdens (Pueschel, Kopito, & Schwachman, 1972). Hair lead levels also vary in relation to the concentration of lead in house-dust (Creason, Hinners, Bumgauner, & Pinkerton, 1975) and in relation to location proximity to a major lead processor (Roberts et al., 1974) and to metallic based pesticides and fertilizers (Hutchinson, et al., 1973).

The fact that subjects came from a mountainous area geographically removed from any urban area suggests that

exposure to environmental lead may be a larger pediatric health problem in rural areas than previously thought. By far the most important documented source of exposure in children has been the ingestion of chips of lead based paint (Center for Disease Control, 1975). Although we have no direct evidence as to the source of the exposure in this study, many of the subjects lived in low-income deteriorating pre-1940 housing, a type commonly found in southern rural areas. These houses, built before titanium oxide began to replace lead pigments in interior paint, were poorly maintained and potential sources of peeling lead paint and broken lead impregnated plaster.

Three other potential sources of exposure warrant discussion. First, illictly distilled whiskey is still widely consumed in the Upper Cumberland Region (Heinrich, 1979). Based on laboratory testing results, Carr (1972) estimated that approximately 40 percent of all illicit whiskey contains some concentration of lead salts. Moreover, lead poisoning resulting from drinking lead contaminated moonshine whiskey is not uncommon. Area public health and school officials reported that moonshine whiskey is sometimes given to children for medicinal purposes by their parents. Additionally, some pregnant mothers in the area were reported to drink moonshine whiskey to alleviate the discomforts of pregnancy, a cultural practice posing a significant prenatal hazard. The idea that lead adversely affects fetal development is hardly a new one,

of course. Lead has long been used in attempts to induce abortion (Rom, 1976). Wibberley and others (1977) found a much higher concentration of lead in placentas from stillborn infants than in infants who survived the neonatal period, and Moore and others (1977) found that retarded children had higher lead levels at two weeks of age than controls. The causative role of illicit whiskey in the retarded children's increased lead burdens is of course mere speculation, since we have no relevant data. The matter is clearly in need of further investigation.

Secondly, most all subjects came from homes where gardens were cultivated, thus raising the issue of exposure through fertilizers and pesticide applications containing lead. Additionally, potatoes are a basic garden staple in the Upper Cumberland Region, and lead arsenite has been used as a defoliant for potatoes in years when the autumn is wet and the halums do not die back adequately. Third, most of the homes used coal for heating and a by product of coal is airborne arsenic. It has been demonstrated that when arsenic and lead occupy the same biological site, arsenic tends to increase the toxicity and absorption of lead (Sittig, 1976). Thus, inhalation of airborne arsenic may have substantially increased the children's lead burdens.

The importance of the above mentioned sources of exposure to undue lead absorption should not be underestimated. The average daily intake of lead from diet and air among young children probably amounts to one-half to two-thirds of

the daily permissible intake, thereby leaving a very narrow margin of safety (King, 1971).

A number of limitations of the study must be considered. First, with no more than 40 children in the mildly retarded group and 27 children in the control group, this is a rather small study and doubts may arise on how representative the findings are, especially in respect to the controls.

Second, the determination of probable cause for retardation from reviewing school records is subject to errors common to retrospective studies in general. However, the most common errors in these studies are those of omission rather than commission. It is unlikely that a school record noting brain injury or a history of rubella is inaccurate; rather the error would occur because of absent data on medically significant factors, e.g., prenatal and perinatal factors. The relative inaccuracy implicit in the identification of the aetiology unknown group, however, probably leads to a lower mean lead concentration in this group, since the unidentified probable cause cases who might be erroneously included would be predicted to show lead concentrations comparable to a normal population. Such a prediction is supported by an earlier study in which a mentally retarded sample with "probable aetiology" showed no significant difference in lead concentrations from those of a normal control sample, while a group of mentally retarded children "aetiology unknown" had statistically significant raised lead concentrations (David, et al., 1976).

The implications of this report are obvious and disturbing. The data shows that elevated lead concentrations in Appalachian children much lower than those associated with overt lead poisoning may also cause neurologic damage albeit a type that is more difficult to define. It is important to reiterate that none of the subjects in this study had lead concentrations associated with lead poisoning.

The findings of this study suggest that a reexamination of lead poisoning concentrations is needed, because concentrations of lead previously thought harmless may now have to be considered lead poisoning and viewed as an aetiological factor in mild mental retardation. Other studies have also recommended a reexamination of lead concentrations, having linked low elevated lead levels to mental retardation, impaired IQ, deficits in visual, fine, and gross motor coordination and hyperactivity--findings previously reported as sequelae of overt lead poisoning (Lin-Fu, 1979).

Finally, it is time to reassess the problem of lead exposure in rural children. The discovery of high hair lead levels among mildly retarded children living in a rural mountainous region seems to go against both common sense and well documented findings concerning the association between undue lead exposure and urban areas. Lead has no known physiologic role in the human body; yet through food, drink, and air, a certain amount is taken in daily. Rural children are further faced with many dangerous sources. Future hazardous exposure must be prevented. As has been historically

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shown in other medical-environmental problems, this may well require an aroused public as a motivating force for meaningful ways of reducing environmental sources of lead exposure.

Table 1 - Demographic Characteristics of 2 Groups of Children

	N	Sex M/F	Age (yr.)		Ethnic Group	
			Mean ± S.D.	Range	Caucasian	Black
Mentally retarded, aetiology unknown	40	29/11	11.00 2.353	4-16	38	2
Control group	27	16/11	11.07 3.198	4-14	26	2

Children in each group came equally from social classes 4 and 5 as defined by Hollingshed and Redlich (1958).

Table 2 - Comparison of Hair-Lead Concentrations Among 2 Groups

Group	N	Hair-lead (ppm.) (mean ± S.D.	T value*
Mentally retarded, aetiology unknown	40	15.90 ± 8.40	3.39**
Control	27	9.16 ± 7.29	

* two tailed
** $p < 0.001$

Table 3 - Distribution of Hair-Lead Concentrations for 2 Groups of Children

Group	Hair-Lead Range (ppm.) - 2-15	15.1-30	30.1 and above
Mentally retarded, aetiology unknown	22	15	3
Control group	22	5	0

15 ppm. is considered the acceptable upper limit of hair lead.

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